Complete Summary

GUIDELINE TITLE

Recommendations for estrogen and progestogen use in peri- and postmenopausal women: October 2004 position statement of The North American Menopause Society.

BIBLIOGRAPHIC SOURCE(S)

Recommendations for estrogen and progestogen use in peri-and postmenopausal women: October 2004 position statement of The North American Menopause Society. Menopause 2004 Nov-Dec;11(6):589-600. [110 references] PubMed

GUIDELINE STATUS

This is the current release of the guideline.

This guideline updates a previous version: Estrogen and progestogen use in peri- and postmenopausal women: September 2003 position statement of The North American Menopause Society. Menopause 2003 Nov-Dec;10(6):497-506.

COMPLETE SUMMARY CONTENT

SCOPE

DISEASE/CONDITION(S)

Menopause-associated symptoms in perimenopausal and postmenopausal women

GUIDELINE CATEGORY

Assessment of Therapeutic Effectiveness
Management

Risk Assessment
Treatment

CLINICAL SPECIALTY

Cardiology
Endocrinology
Family Practice
Geriatrics
Internal Medicine
Obstetrics and Gynecology
Oncology

INTENDED USERS

Health Care Providers
Physician Assistants
Physicians

GUIDELINE OBJECTIVE(S)

To present clinical recommendations for use of hormone therapy (HT) in peri- and postmenopausal women

TARGET POPULATION

Peri- and postmenopausal women

INTERVENTIONS AND PRACTICES CONSIDERED

Menopause-related hormone therapy, including:

1. Estrogen therapy (ET)
2. Systemic ET/estrogen-progestogen therapy (EPT)
3. Local ET
4. Progestogen therapy (progesterone and progestin)

MAJOR OUTCOMES CONSIDERED

The risk-benefit ratio of postmenopausal estrogen therapy (ET) and estrogen-progestogen therapy (EPT) for both disease prevention and treatment of specific menopause-related symptoms

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

The 2004 Panel utilized the 2002 and 2003 reports as a starting point. A comprehensive literature search was conducted to identify all new papers published subsequent to the 2003 report. Panelists also submitted relevant papers.

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

The level of evidence indicated for each study is based on a grading system that evaluates the scientific rigor of the study design, as developed by the U.S. Preventive Services Task Force.

Levels of Evidence

Level I: Properly randomized, controlled trial

Level II-1: Well-designed controlled trial but without randomization

Level II-2: Well-designed cohort or case-control analytic study, preferably from more than one center or research group

Level II-3: Multiple time series with or without the intervention (e.g., cross-sectional and uncontrolled investigational studies); uncontrolled experiments with dramatic results could also be regarded as this type of evidence.

Level III: Opinions of respected authorities that are based on clinical experience; descriptive studies and case reports; reports from expert committees

METHODS USED TO ANALYZE THE EVIDENCE

Review
Review of Published Meta-Analyses

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

The North American Menopause Society (NAMS) Board of Trustees convened a third Hormone Therapy Advisory Panel to develop an updated report on hormone therapy.

The 2004 Panel utilized the 2002 and 2003 reports as a starting point. Considering all the evidence, Panelists were asked to provide their current view of all items of consensus and nonconsensus from the 2003 report. Each Panelist provided comments independently (i.e., unaware of the responses of the other Panelists). All responses were collated in the North American Menopause Society's Central Office into two lists: those with consensus and those without. All responses were distributed to the entire Panel. The Panel reviewed all of the responses by telephone conference call in an attempt to reach consensus. Further development of the report through multiple drafts was conducted through the Internet. The clinical recommendations indicate where consensus was achieved as well as where opinions differed.

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Not applicable

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Internal Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

The position statement was reviewed and approved by The North American Menopause Society (NAMS) 2003-2004 Board of Trustees.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

Recommendations for Clinical Practice: Areas of Consensus

The Panel agreed on the following clinical recommendations for postmenopausal hormone therapy:

- A strong recommendation was made for uniform and consistent terminology for menopause-related therapies, as indicated below:

  ET: Estrogen therapy

EPT: Combined estrogen-progestogen therapy

HT: Hormone therapy (encompassing both ET and EPT)

CC-EPT: Continuous-combined estrogen-progestogen therapy (daily administration of both estrogen and progestogen)

CS-EPT: Continuous-sequential estrogen-progestogen therapy (estrogen daily, with progestogen added on a set sequence)

Systemic ET/EPT: Preparations of ET or EPT that have a systemic, not solely vaginal, effect

Local ET: Preparations of ET that have a predominately vaginal, not systemic, effect

Progestogen: Encompassing both progesterone and progestin

- Treatment of moderate to severe menopause symptoms (i.e., vasomotor symptoms, sleep disruption from vasomotor symptoms) remains the primary indication for systemic ET and EPT. Every systemic ET/EPT product is government approved for this indication.
- Every systemic and local ET/EPT product is government-approved for treating moderate to severe symptoms of vulvar and vaginal atrophy, such as vaginal dryness, dyspareunia, and atrophic vaginitis. When hormones are considered solely for this indication, local ET is generally recommended.
- The primary menopause-related indication for progestogen use is endometrial protection from unopposed ET. For all women with an intact uterus who are using estrogen therapy, clinicians are advised to prescribe adequate progestogen, in either a CC-EPT or CS-EPT regimen. Postmenopausal women without a uterus should not be prescribed a progestogen.
- Some women with an intact uterus who choose EPT may experience undesirable side effects from the progestogen component. However, there is insufficient evidence regarding endometrial safety to recommend use of long-cycle progestogen (i.e., progestogen every 3-6 months for 12-14 days), a progestin-containing intrauterine device (IUD), or low-dose estrogen without progestogen as an alternative to standard EPT regimens. If utilizing any of these approaches, close surveillance of the endometrium is recommended, pending more definitive research. There are encouraging data on the efficacy of lower-dose therapies with reduction of side effects.
- ET and EPT did not reduce coronary heart disease (CHD) incidence in the Women's Health Initiative (WHI) study. The role of ET/EPT in primary prevention of CHD remains unclear when considered for peri- and early postmenopausal women if started early and continued for a number of years, and needs further evaluation. Until that evidence is forthcoming, no ET or EPT regimen should be used for primary or secondary prevention of CHD.
- ET and EPT may increase the risk of ischemic stroke in postmenopausal women, but randomized controlled trial data are not consistent in this regard. The WHI EPT and ET arms demonstrated an increased risk while other large trials have not. The attributable absolute increased risk of stroke based on WHI data, under the Council for International Organizations of Medical Sciences (CIOMS) classification, falls into the rare category. The Panel

concluded that no HT regimen should be used for primary stroke prevention. In women with a history of CHD or ischemic cerebrovascular disease, ET does not significantly influence stroke risk (secondary prevention). It is therefore important to reduce the risk of stroke regardless of HT use in these women.

- Breast cancer risk probably increases with EPT use beyond 5 years. In absolute terms, this increased risk is small in the WHI, being 4 to 6 additional invasive cancers per 10,000 women who use it for 5 or more years and of possible statistical significance. There is no mortality difference between EPT users and nonusers. Studies have not clarified whether the risk differs between continuous or sequential use of progestogen. Women in the estrogen-only (CEE) arm of the WHI demonstrated no increase in risk of breast cancer after an average of 6.8 years of use, and there was a nonsignificant trend toward reduction of breast cancer in women overall, with this trend strongest in women under age 60 (7 fewer breast cancers per 10,000 women using ET). Available evidence also suggests that estrogen alone for fewer than 5 years has little impact on breast cancer risk, although this question persists despite the WHI results. A large observational study has shown that 25 years of ET use is not associated with breast cancer risk. Specific subgroups may be affected in different ways. There are no substantial data reporting any increase in mortality with HT. EPT and, to a lesser extent, ET increase breast cell proliferation, breast pain, and mammographic density, and EPT may impede the diagnostic interpretation of mammograms. Evidence suggests that unopposed CEE is unlikely to have a significant effect on mammography.
- There is definitive evidence for ET and EPT efficacy in reducing risk for postmenopausal osteoporosis fracture. Many EPT and ET products are government-approved for prevention of postmenopausal osteoporosis (i.e., loss of bone mineral density) through long-term treatment. For women who require drug therapy for osteoporosis risk reduction (including women at high risk of fracture in the next 5-10 years), ET/EPT can still be considered, weighing its risks and benefits as well as those of alternate therapies.
- Initiating EPT after age 65 should not be recommended for primary prevention of dementia as it may increase the risk of dementia during the ensuing 5 years in this population. The evidence is insufficient to either support or refute the efficacy or harm of ET/EPT for primary prevention of dementia when therapy is initiated during the menopause transition or early postmenopause. ET does not appear to convey direct benefit or harm for treatment of dementia due to Alzheimer's disease.
- The effects of ET/EPT on risk for breast cancer and osteoporotic fracture in perimenopausal women with moderate to severe menopause symptoms have not been established in randomized clinical trials. The findings from trials in different populations (e.g., WHI) should, therefore, be extrapolated with caution.
- Data from studies such as the WHI and the Heart and Estrogen/progestin Replacement Study (HERS) should not necessarily be extrapolated to symptomatic postmenopausal women younger than 50 years of age who initiate HT, as these women were not studied in these trials. WHI and HERS involved predominantly asymptomatic postmenopausal women aged 50 and over (with mean ages of 63 and 67, respectively), the majority of whom were 10 years or more beyond menopause, and HERS was conducted solely among women with known coronary artery disease. The data should not be extrapolated to women experiencing premature menopause ($\leq$40 years of age) and initiating HT at that time.

- Premature menopause and premature ovarian failure are conditions associated with earlier onset of osteoporosis and CHD, but there are no clear data as to whether ET or EPT will reduce morbidity or mortality from these conditions. The benefit-risk ratio may be more favorable for younger women who initiate therapy at an early age.
- Use of ET and EPT should be consistent with treatment goals, benefits, and risks for the individual woman, taking into account symptoms and domains (e.g., sexuality, sleep) that may have an impact on quality of life.
- Lower-than-standard doses of ET and EPT should be considered (i.e., daily doses of 0.3 mg oral conjugated estrogens, 0.25 to 0.5 mg oral micronized 17beta-estradiol, 0.025 mg 17beta-estradiol patch, or the equivalent). Many studies have demonstrated nearly equivalent vasomotor and vulvovaginal symptom relief and preservation of bone mineral density. However, some women may need additional local therapy for persistent vaginal symptoms. Lower ET and EPT doses are better tolerated and may have a better benefit-risk profile than standard doses. However, lower doses have not been tested in long-term trials.
- Nonoral routes of administration of ET/EPT may offer advantages and disadvantages, but the long-term benefit-risk ratio has not been demonstrated. Differences would be related to the role of the first-pass hepatic effect, the hormone concentrations in the blood achieved by a given route, and the biologic activity of active component ingredients. There is some evidence that transdermal 17beta-estradiol may be associated with lower risk of deep venous thrombosis than oral estrogen and to a non-significant increase in CHD risk relative to placebo. A large observational study has shown similar increased risks for breast cancer with both oral and transdermal estrogens.
- Extended use of the lowest effective dose for treatment goals of ET or EPT is acceptable under the following circumstances, provided the woman is well aware of the potential risks and benefits and that there is clinical supervision:
    - For the woman for whom, in her opinion, benefits of menopause symptom relief outweigh risks, notably after failing an attempt to withdraw HT
    - For women who are at high risk for osteoporotic fracture and also have moderate to severe menopause symptoms
    - For further prevention of bone loss in women with established reduction in bone mass when alternate therapies are not appropriate for that woman or cause side effects or when the outcomes of the extended use of alternate therapies are unknown.
- Prior to consideration of any therapeutic regimen, including ET/EPT, all women should have a complete health evaluation, including a comprehensive history, physical examination, and mammography. Other specific examinations, such as bone densitometry, should be considered on a case-by-case basis.
- The Panel concluded that with regard to duration of use, a general guiding principle should be for the lowest effective dose and time consistent with treatment goals. The Panel recognized that symptoms can recur when therapy is discontinued, independent of age and duration of ET/EPT use. The Panel agreed that the decision to continue HT should be individualized based on severity of symptoms, current risk-benefit considerations, and that the woman in consultation with her health-care provider believed that continuation of therapy is warranted.

- The Panel concluded that an improvement in health-related quality of life (HQOL) can result through decreased menopause symptoms and possible elevation of mood that leads to a feeling of well-being. There is a lack of consensus on the impact of HT on overall quality of life (QOL) and health-related quality of life in asymptomatic women. In part this is due to a lack of agreement regarding how best to obtain an appropriate evaluation of quality of life in women after menopause, including the domains to be incorporated into any survey instruments. There is consensus that validated instruments for determining the impact of HT, or indeed any menopause-related therapy, on both overall quality of life and health-related quality of life should be incorporated into future studies.
- The Panel recognized that specific compounds, dose, and route of administration may have different outcomes. Nonetheless, in the absence of clinical trial data for each specific product, the clinical trial results for one agent should be generalized to all agents within the same family. This proviso also applies to the so-called bioidentical products.

Areas Where Insufficient or Conflicting Evidence Precludes Consensus

The Panel could not reach consensus on the following issues, but the summary of responses is of relevance to clinicians:

Is HT associated with early risk of CHD?

Panelists were divided on the issue as to whether there is definitive evidence for early increased risk of CHD with HT. For women similar to participants in the EPT arm of WHI (average age 63 years; range from 50 to 79 years), the WHI data are the best estimate of early harm from EPT. The WHI demonstrated that EPT may increase the risk of CHD during the first year of hormone use among generally healthy postmenopausal women in whom HT is initiated up to 20 or more years after menopause. The attributable risk in this instance, under the CIOMS classification, falls into the rare category. In addition, in HERS the increased risk of CHD in the first year due to EPT use was not observed in women who were concomitantly using statin therapy. There is also evidence that early harm within the first year of use may not pertain to healthy postmenopausal women using ET/EPT for menopause symptom management. Increased risk of CHD in the first year was not observed in the ET arm of the WHI or in any other ET-only study.

Should women who are doing well on long-term HT discontinue?

The Panelists were divided in opinion as to whether women on well established long-term therapy should be advised to discontinue at a specific duration of therapy. No recommendation is made, but there is agreement that the risks and benefits must be discussed on an individualized basis.

Is there a best way to discontinue HT?

When a decision is made to discontinue therapy, Panelists were divided in their recommendations regarding abrupt therapy cessation versus tapering the dose. Past history of severe symptoms may favor tapering, but no specific protocols could be recommended. Some gradually decrease the dose, while others lengthen the time between doses. Matrix transdermal HT patches can be trimmed to provide smaller doses. Current data are inadequate to suggest that one method is better than the other.

Does a continuous-combined EPT regimen (CC-EPT) have an effect different from continuous estrogen with sequential progestogen (CS-EPT)?
There are some indications that continuous progestogen in the dosages administered in studies such as the WHI and HERS may be related to these trials' adverse breast cancer and cardiovascular outcomes, but conflicting data preclude a consensus.

CLINICAL ALGORITHM(S)

None provided

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The position statement was supported by evidence from randomized, controlled clinical trials, meta-analyses, and review articles. If the evidence was contradictory or inadequate to form a conclusion, a consensus-based opinion was made.

A list of the most current references regarding hormone therapy (HT) use is provided at the end of the original guideline document. The level of evidence indicated for each study is based on a grading system that evaluates the scientific rigor of the study design, as developed by the United States Preventive Services Task Force. See "Rating Scheme for the Strength of the Evidence" for definitions of these levels.

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

Overall Benefits

- Appropriate use of hormone therapy (HT) in peri- and postmenopausal women to maximize health benefits while minimizing health risks
- Relief of moderate to severe menopause symptoms

Specific Benefits

There is definitive evidence for combined estrogen-progestogen therapy (EPT) efficacy in reducing risk for postmenopausal osteoporosis fracture.

Subgroups Most Likely to Benefit

- Women with moderate to severe menopause symptoms who are at high risk for osteoporotic fracture
- High-risk women for whom alternate therapies for the prevention of osteoporosis are not appropriate

POTENTIAL HARMS

- Breast cancer risk probably increases with combined estrogen-progestogen therapy (EPT) use beyond 5 years.
- Estrogen-progestogen therapy and, to a lesser extent, estrogen therapy increase breast cell proliferation, breast pain, and mammographic density, and estrogen-progestogen therapy may impede the diagnostic interpretation of mammograms.

QUALIFYING STATEMENTS

QUALIFYING STATEMENTS

This position statement focuses on the use of government-approved prescription estrogen therapy/combined estrogen-progestogen therapy (ET/EPT) products available in the United States and Canada, not custom estrogen therapy/combined estrogen-progestogen therapy preparations, selective estrogen-receptor modulators (SERMs), or hormones available without a prescription (including phytoestrogens).

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better
Staying Healthy

IOM DOMAIN

Effectiveness
Patient-centeredness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

Recommendations for estrogen and progestogen use in peri-and postmenopausal women: October 2004 position statement of The North American Menopause Society. Menopause 2004 Nov-Dec;11(6):589-600. [110 references] PubMed

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

2002 Oct 6 (revised 2004 Oct 6)

GUIDELINE DEVELOPER(S)

The North American Menopause Society - Private Nonprofit Organization

SOURCE(S) OF FUNDING

The North American Menopause Society (NAMS)

GUIDELINE COMMITTEE

Hormone Therapy Advisory Panel

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

The Panel was composed of acknowledged clinical and research experts (both NAMS members and nonmembers) from relevant areas of menopause-related health-care.

Advisory Panel Members

Wulf H. Utian, MD, PhD, FACOG (Chair) - The Arthur H. Bill Professor Emeritus of Reproductive Biology and Obstetrics and Gynecology, Case Western Reserve University School of Medicine; Consultant in Gynecology, The Cleveland Clinic Foundation; President, Rapid Medical Research Inc., Cleveland, OH; NAMS Executive Director and Honorary Founding President, NAMS President 1989-1992, NAMS Board of Trustees 1989-Present

David F. Archer, MD - Professor of Obstetrics and Gynecology, Eastern Virginia Medical School, Norfolk, Virginia; NAMS President 1997-1998, NAMS Board of Trustees 1995-2000

J. Chris Gallagher, MD - Professor of Medicine, Department of Endocrinology and Metabolism, Creighton University Medical School, Omaha, NE; NAMS President 1994-1995, NAMS Board of Trustees 1990-1996 and 2002-Present

Margery L.S. Gass, MD - Professor of Clinical Obstetrics and Gynecology, University of Cincinnati College of Medicine; Director, University Hospital Menopause and Osteoporosis Center, Cincinnati, OH; NAMS 2002-2003 President, NAMS Board of Trustees 1999-Present; WHI and WHIMS Investigator

Morrie M. Gelfand, CM, MD - Professor of Obstetrics and Gynecology, McGill University; Honorary Chief of Obstetrics and Gynecology, The Sir Mortimer B. Davis Jewish General Hospital; Co-Director, Mc-Gill University Menopause Clinic, Montreal, QC, Canada; NAMS 2001-2002 President, NAMS Board of Trustees 1997-2003

Victor W. Henderson, MD, MS - Professor of Epidemiology and Neurology, Stanford University School of Medicine, Palo Alto, CA; NAMS Board of Trustees 2002-Present; Member, WHIMS External Advisory Board

Howard N. Hodis, MD - Professor of Medicine and Preventive Medicine and Molecular Pharmacology and Toxicology; Director, Atherosclerosis Research Unit, Division of Cardiovascular Medicine, University of Southern California Keck School of Medicine, Los Angeles, CA

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Marcia L. Stefanick, PhD, FAHA - Professor of Medicine, and Obstetrics and Gynecology, Stanford University, Palo Alto, CA; HERS, WHI, and WHIMS Investigator, Chair, WHI Steering Committee

Nancy Fugate Woods, PhD, RN, FAAN - Dean, School of Nursing, Professor, Family and Child Nursing, University of Washington, Seattle, WA; NAMS President 1999-2000, NAMS Board of Trustees 1997- 2002; WHI Investigator

FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

Panelist Disclosures

Chair, Wulf H. Utian, MD, PhD, FACOG - Industry consulting fees: Berlex, Eli Lilly, Endeavor, GSK, Johnson & Johnson Pharmaceutical Research, Merck-Theramex, Pfizer, Roche, Warner Chilcott. Direct industry lecture fees: None disclosed. Industry research support: Amylin, 3M, Barr, Berlex, Bristol-Myers Squibb, Eli Lilly, Endeavor, Forest, Galen, GlaxoSmithKline, Neurocrine Biosciences, Novartis, Novo Nordisk, Organon, Pfizer, Pharmacia, Procter & Gamble, Roche, Sepracor, Solvay, Wyeth, Yamanouchi.

David F. Archer, MD - Industry consulting fees: Agile Therapeutics, Berlex, Endeavor, Genentech, Galen, Lilly, Novo Nordisk, Organon, Schering, Solvay, Watson, Wyeth. Direct industry lecture fees: Berlex, Novo Nordisk, Solvay, Wyeth. Industry research support: Amylin, Barr, Berlex, Galen, Insmed, Lilly, Organon, Parke-Davis, Pharmacia, Solvay, Wyeth, Yamanouchi.

J. Chris Gallagher MD - Industry consulting fees: Aventis, Endeavor, Lilly, Pfizer, Roche, Wyeth. Direct industry lecture fees: Aventis, Organon, Pfizer, Roche, Wyeth. Industry research support: Endeavor, Organon, Pfizer, Roche, Solae, Wyeth.

Margery L.S. Gass, MD - Industry consulting fees: Eli Lilly, GlaxoSmithKline, Merck, Procter & Gamble. Direct industry lecture fees: Aventis. Industry research support: Eli Lilly, Glaxo- SmithKline, Merck, Pfizer, Procter & Gamble, Wyeth.

Morrie M. Gelfand, CM, MD - Industry consulting fees: Procter & Gamble. Industry lecture fees: None disclosed. Industry research support: Pfizer.

Victor W. Henderson, MD, MS - Industry consulting fees: None disclosed. Direct industry lecture fees: None disclosed. Industry research support: None disclosed.

Howard N. Hodis, MD - Industry consulting fees: None disclosed. Direct industry lecture fees: None disclosed. Industry research support: None disclosed.

Rogerio A. Lobo, MD - Industry consulting fees: Berlex, Merck, Novartis, Ortho-McNeil, Pfizer, Solvay, Wyeth. Direct industry lecture fees: None disclosed. Industry research support: Novartis, Wyeth.

Michael McClung, MD - Industry consulting fees: Amgen, Aventis, Lilly, Merck, Novartis, NPS, Pfizer, Procter & Gamble, Roche, Wyeth. Direct industry lecture fees: Aventis, Merck. Industry research support: Amgen, Aventis, Lilly, Merck, Novartis, NPS, Pfizer, Procter & Gamble, Roche.

Robert L. Reid, MD - Industry consulting fees: Lilly Canada. Direct industry lecture fees: Wyeth Canada. Industry research support: Wyeth.

Peter E. Schwartz, MD - Industry consulting fees: None disclosed. Direct industry lecture fees: None disclosed. Industry research support: None disclosed.

Marcia L. Stefanick, PhD - Industry consulting fees: None disclosed. Direct industry lecture fees: None disclosed. Industry research support: None disclosed.

Nancy Fugate Woods, PhD, RN, FAAN - Industry consulting fees: None disclosed. Direct industry lecture fees: None disclosed. Industry research support: None disclosed.

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GUIDELINE AVAILABILITY

Electronic copies: Available in Portable Document Format (PDF) from The North American Menopause Society (NAMS) Web site.

Print copies: Available from NAMS, P.O. Box 94527, Cleveland, OH 44101, USA Order forms are available at The North American Menopause Society [NAMS] Web site, www.menopause.org

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

This NGC summary was completed by ECRI on January 23, 2003. The information was verified by the guideline developer on February 13, 2003. This summary was updated by ECRI on March 5, 2004. The information was verified by the guideline developer on March 29, 2004. This NGC summary was updated by ECRI on December 29, 2004. The updated information was verified by the guideline developer on January 24, 2005.

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